Evidence has been reported that ACTH does not hasten the recovery of the normal pituitary-adrenal function in patients who experienced prolonged steroid-induced pituitary-adrenal suppression.

Prolonged ACTH therapy has been shown to produce antibodies to ACTH that cross-react with endogenous ACTH, binding it in the circulation in inactive form.

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The Management of Respiratory Failure In Childhood Status Asthmaticus

In the management of 30 episodes, the criteria for respiratory failure consisted of the following clinical signs: decreased or absent inspiratory breath sounds, severe inspiratory retractions and use of accessory muscles, cyanosis in 40 percent oxygen, depressed level of consciousness and poor skeletal muscle tone.

The technique evolved included tracheal intubation followed by general anesthesia and manual ventilation, mechanically assisted ventilation with a pressure-flow cycled ventilator under heavy sedation, neuromuscular blockade with d-tubocurarine and light sedation and controlled ventilation with the Emerson volume regulated respirator.

Blood gases were determined frequently. There were 18 complications, including one death. The investigators reported an experienced, constantly available team is necessary.

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Hypoxia in Asthmatic Attacks

The dehydrated adrenalin-fast patient is acutely ill and needs intensive care management. All such patients have hypoxia, and arterial blood gas measurements are essential in control to prevent respiratory failure.

A pCO_2 of 60 or above is an indication of the need for assisted ventilation by means of a Bennett, Bird or Emerson machine with the use of either a laryngeal catheter or tracheotomy. A pO_2 below 50 increases danger of cardiac arrest. Oxygen can be given at a low flow rate as frequent pCO_2 determinations are done.

Steroids in large doses and antibiotics are also essential to proper management.

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Aspirin Sensitivity

Untoward reactions to aspirin take two forms: (a) appearance of the expected symptoms of over-dosage from normally tolerated amounts, and (b) the more serious allergic or anaphylactic type of sensitivity. In children, sensitivity usually appears in the form of urticaria, angio-edema or a macular eruption, occasionally with purpura. Sensitivity in adults tends to appear in middle life—in my experience, most often in women between 35 and 45 years of age. The typical patient has suffered from a vasomotor rhinitis leading to nasal polyposis. Only 10 percent show evidence of atopy, or familial allergy. An "intrinsic" (non-allergic) asthma may precede or coincide with the onset of aspirin sensitivity. The asthma usually follows a respiratory infection, but may appear suddenly after nasal operations. Once aspirin sensitivity is established, even minute amounts of aspirin can produce alarming or even fatal bronchospasm.

The sensitivity is highly specific: among the salicylates only acetyl salicylic acid provokes the reaction. However, cross reactions regularly occur

with indomethacin, aminopyrine and antipyrine; to a lesser extent with FD+C Yellow No. 5 food coloring.

Acetyl salicylic acid appears not to be an antigen or antigenic determinant in the usual sense, and no specific antibody has ever been found. Samter and Beers postulated that in these patients aspirin potentiates rather than inhibits the activity of the kinin receptors in skin, nasal membranes and bronchioles.

The asthma that accompanies aspirin sensitivity is usually difficult to control, and can lead more or less rapidly to decided pulmonary insufficiency. The nasal polyps recur regularly after removal. Generally the best method of handling the aspirin sensitivity-asthma-nasal polyposis triad consists of prompt and vigorous treatment of respiratory infections with broad spectrum antibiotics, systematic use of oral bronchodilators and, from time to time, vasoconstrictor-antihistamine combinations by mouth. Small amounts of prednisone or prednisolone given daily or intermittently can be remarkably effective for long periods.

In patients with demonstrable atopic allergy, careful immunotherapy can reduce the frequency and severity of the asthma, and retard (if not prevent) regrowth of nasal polyps.

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Pathology of Bronchial Obstruction In Asthma

Bronchial asthma is a chronic disease characterized by paroxysmal bronchial obstruction and ventilatory insufficiency. Bronchi and bronchioles are the seat of the essential pathological changes. There is mucosal edema, hypersecretion of a thick tenacious mucus and smooth muscle contractions. Extrinsic asthma occurs in individuals who have been sensitized followed by a reexposure of the specific antigen. An antigen-antibody reaction occurs in a lung followed by release of histamine, slow-reacting substance of anaphylaxis, bradykinin, and other substances which are pharmacologically active. The action of these substances on mucous glands, smooth muscle and blood vessels presumably produces the asthma. The obstruction to airflow caused by mucosal edema, mucus hypersecretion and smooth muscle spasm results in a decided narrowing of the bronchial tree.

In the severe forms the obstruction causes hypoxemia, hypercapnia, cor pulmonale and finally death from widespread obstruction of the airways by inspissated mucus. The mucus is thick and stringy, causes coughing and contributes to the wheezing and the shortness of breath. As time goes on the smooth muscle becomes thickened, the mucous glands become prominent, and distended mucus-engorged goblet cells are common in the bronchiolar wall. There is thickening of the basement membrane. At autopsy a cellular infiltration is found that consists mainly of eonsinophils and frequently plasma cells. Additional pulmonary lesions which are due to complications from the above are chronic bronchitis, atelectasis and peribronchial and pulmonary fibrosis.

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Life-Threatening Asthma

Errors which may contribute to respiratory failure in the treatment of life-threatening asthma are improper use of sedation or oxygen, or inadequate steroid administration.

Decreasing wheezing by auscultation despite increasing dispnea is an ominous sign of impending respiratory failure. Therapy should include antiasthmatic medication, antibiotics and proper use of oxygen and steroids. The decision as to when to start assisted ventilation can be made on the basis of blood gas values and clinical judgment. If the pCO_2 is rising while the pO_2 and pH are falling despite active treatment, assisted ventilation may be needed. Three patients were ventilated by use of intermittent positive pressure breathing and five required a volume respirator.

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